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ADDITION OF SPECIFIC LIPOPROTEIN(A) APHERESIS IN STABLE ISCHEMIC HEART DISEASE PATIENTS CONTRIBUTES TO CAROTID ATHEROSCLEROSIS PREVENTION

Poster Contributions
Poster Hall B1
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Session Title: Lipids, Novel Therapies and Acute Coronary Syndromes
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Background: The purpose of this study was to evaluate the effect of 18-month course of specific lipoprotein(a) (Lp(a)) apheresis on carotid intima-media thickness (CIMT) progression in patients with controlled low-density lipoprotein cholesterol (LDL-C) levels reached with atorvastatin treatment, but with high Lp(a) levels, during 42 months of follow-up.

Methods: In this single-center, partially-blind study 30 stable ischemic heart disease patients with Lp(a) level ≥50 mg/dL on a guideline-driven therapy were assigned to Lp(a) immunoadsorption columns (n=15, Lp(a) Lipopak®, POCARD Ltd., Russia) on a weekly basis or atorvastatin monotherapy after a 4-week diet and atorvastatin run-in period. The primary efficacy end-point was the absolute change from baseline through 18 months in mean of left and right CIMT, as assessed by duplex ultrasound scanning.

Results: As the only target for the specific Lp(a) apheresis, Lp(a) level decreased by an average of 73±12% (a mean for 951 procedures) to 29±16 mg/dL immediately after the session. During 18 months of regular treatment, the interval mean values for Lp(a) were 73.3±13.2 mg/dL. The final change in Lp(a) level in the immunoadsoption group was -31.7±22.3 mg/dL, as compared with 4.8±10.8 mg/dL in the atorvastatin only group (P <0.0001). LDL-C level corrected for Lp(a) cholesterol unchanged: -7.7 ± 23.2 and -3.9 ± 23.2 mg/dL for apheresis and control groups, respectively (P=0.65). At 18 months the primary efficacy end point decreased by -0.07±0.15 mm (P=0.01) in the Lp(a) apheresis group and by -0.03±0.13 (P=0.09) in the control group. Two years after apheresis termination, in the immunoadsorption-treated patients (n=14) Lp(a) level was 103±23 mg/dL and CIMT increased in average by 0.02±0.08 mm (P=0.12 in comparison with the baseline). In the control group Lp(a) was 101±52 mg/dL and CIMT increased by up to 0.06±0.10 mm (P=0.18 versus baseline, P=0.03 for between-group comparison).

Conclusion: Specific Lp(a) apheresis during 18 months resulted in significant regression of CIMT in comparison with the traditional approach despite achieved LDL-C levels in both groups. Lp(a) lowering strategy led to long-term stabilization in the rate of CIMT progression.